Early Journal Content on JSTOR, Free to Anyone in the World

This article is one of nearly 500,000 scholarly works digitized and made freely available to everyone in the world by JSTOR.

Known as the Early Journal Content, this set of works include research articles, news, letters, and other writings published in more than 200 of the oldest leading academic journals. The works date from the mid-seventeenth to the early twentieth centuries.

We encourage people to read and share the Early Journal Content openly and to tell others that this resource exists. People may post this content online or redistribute in any way for non-commercial purposes.

Read more about Early Journal Content at http://about.jstor.org/participate-jstor/individuals/early-journal-content.

JSTOR is a digital library of academic journals, books, and primary source objects. JSTOR helps people discover, use, and build upon a wide range of content through a powerful research and teaching platform, and preserves this content for future generations. JSTOR is part of ITHAKA, a not-for-profit organization that also includes Ithaka S+R and Portico. For more information about JSTOR, please contact support@jstor.org.

THE BIOLOGY OF DEATH—V. THE INHERITANCE OF DURATION OF LIFE IN MAN[1]

By Professor RAYMOND PEARL
THE JOHNS HOPKINS UNIVERSITY

1. The Determination of Longevity

IN the series of papers up to this point we have seen, in the first place, that immortality is a potential attribute of cells generally and becomes actually realized when conditions are so arranged as to make continued life possible. These conditions are not realized in the metazoan body because of differentiation and specialization of structure and function. What actually happens in the metazoa is that sooner or later some differentiated organ or system of organs gets to functioning badly and upsets the delicate balance of the whole. As a result the entire organism presently dies. We have further seen that in the case of man, where alone quantitative data are available, the breakdown of particular organ systems, and consequent death of the whole, occurs in a highly orderly manner in respect of time or age. Each organ system has a characteristic time curve for its breakdown, differing from the curve of any other system. The problem which now confronts us is to find out what lies back of these characteristic time curves and determines their form. In view of the biological facts about death which we have learned, what determines that John Smith shall die at 58, while Henry Jones lives to the obviously more respectable age of 85? We have seen that there is every reason to believe that all the essential cells of both their bodies are inherently capable under proper conditions of living indefinitely. We are further agreed that it is the differentiated and specialized structure of their bodies which prevents the realization of these favorable conditions. But all this helps us not at all to understand why in fact one lives nearly 30 years longer than the other.

It may help to visualize this problem of the determination of longevity to consider an illustrative analogy. Men behave in respect of their duration of life not unlike a lot of eight-day clocks cared for by an unsystematic person, who does not wind them all to an equal degree and is not careful about guarding them from accident. Some he winds up fully, and they run their full eight days. Others he winds only half way and they stop after four days. Again the clock which has been wound up for the full eight days may fall off the shelf and be brought to a stop at the third day. Or someone may throw some sand in the works when the caretaker is off his guard. So, similarly, some men behave as though they had been wound up for a full 90-year run, while others are but partially wound up and stop at 40 or 65, or some other point. Or, again, the man wound up for 80 years may, like the clock, be brought up much short of that by an accidental invasion of microbes, playing the rôle of the sand in the works of the clock. It is of no avail for either the clock or the man to say that the elements of the mechanism are in whole or in major part capable of further service. The essential problem is: what determines the goodness of the original winding? And what relative part do external things play in bringing the running to an end before the time which the original winding was good for? It is with this problem of the winding up and running of the human mechanism that the present paper will deal.

---

[1] Papers from the Department of Biometry and Vital Statistics. School of Hygiene and Public Health, Johns Hopkins University, No. 32.

There are two general classes of factors which may be involved here. These are, on the one hand, heredity and, on the other hand, environment, using the latter term in the broadest sense. Inasmuch as we can be reasonably sure on *a priori* grounds that longevity, like most other biological phenomena, is influenced by both heredity and environment, the problem practically reduces itself to the measuring of the relative importance of each of these two factor groups in determining the results we see. But before we start the discussion of exact measurements in this field, let us first examine some of the general evidence that heredity plays any part at all in the determination of longevity.

2. THE HYDE FAMILY

The first material which we shall discuss is that provided by the distinguished eugenist, Dr. Alexander Graham Bell, in his study of the Hyde family. Every genealogist is familiar with the "Genealogy of the Hyde Family," by Reuben H. Walworth. It is one of the finest examples in existence of careful and painstaking genealogical research. Upon the data included in this book, Bell has made a most interesting and penetrating analysis of the factors influencing longevity. At first thought one might conclude that highly biased results would probably flow from the consideration of only one family. Bell meets this point very well, however, in the following words:

> A little consideration will show that the descendants did not constitute a single family at all, and indeed had very little of the Hyde blood in them.
>
> Even the children of William Hyde owed only half of their blood to him, and one-half to his wife. The grandchildren owed only one-quarter of their blood to William Hyde, and three quarters to other people, etc. The descendants of the seventh generation, and there are hundreds of them, owed only one sixty-fourth of their blood to William Hyde, and sixty-three sixty-fourths to the new blood introduced through successive generations of marriages with persons not of the Hyde blood at all.

It will thus be seen that the thousands of descendants noted in the Hyde Genealogy constitute rather a sample of the general population of the country than a sample of a particular family in which family traits might be expected to make their appearance.

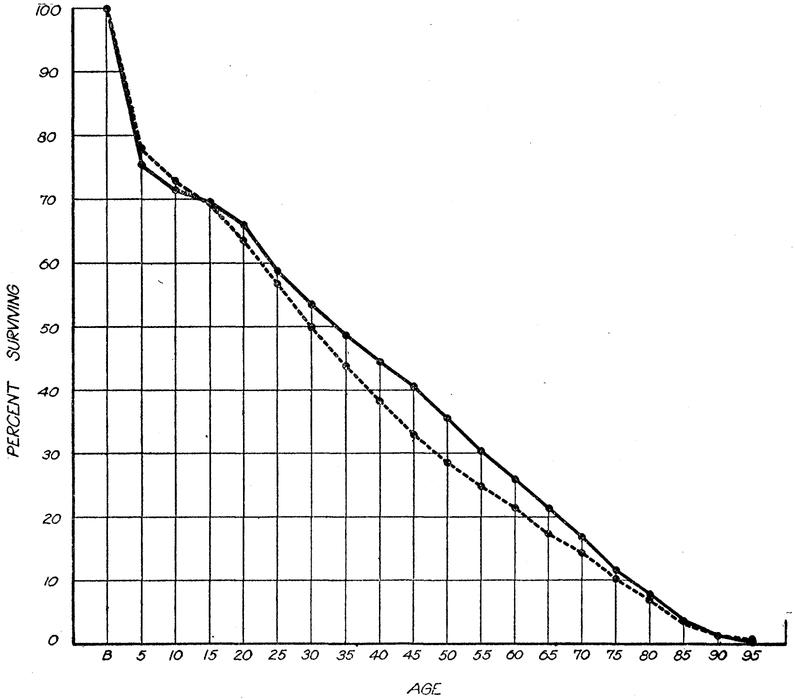

FIG. 1. SHOWING SURVIVAL CURVES OF MEMBERS OF THE HYDE FAMILY
(Plotted from Bell's data)

The substantial normality of the material is shown in Figure 1, which gives the l_x line, that is, the number of survivors at each age, of the 1,606 males and 1,352 females for whom data were available. The solid line is the male l_x line and the dotted line the female l_x. It is at once apparent that the curves have the same general sweep in their passage over the span of life as has the general population life curve discussed in the preceding paper. The descent is a little steeper in early adult life. The female curve differs in two respects from the normal general population curves. In the first place, beginning at age 15 and continuing to age 90, the female curve lies below that for the males, whereas normally for the general population it lies above it. This denotes a shorter average duration of life in the females than in the males, the actual figures being 35.8 years for the males and 33.4 years for the females. Bell attributes the difference to the strain of child-bearing by the females in this rather highly fertile group of people, belonging in the main to a period when restrictions upon size of family were less common and less extensive than now. In the second place, the female l_x curve is actually convex to the base through-out a considerable portion of middle life whereas normally this portion of the curve presents a concave face to the base.

Apart from these deviations, which are of no particular significance for the use which Bell makes of the data, the Hyde material is essentially normal and similar to what one would expect to find in a random sample of the general population. In this material there were 2,287 cases in which the ages at death of the persons and the ages at death of their fathers were known. It occurred to Bell to arrange this material in such a way as to show what, if any, relation existed between age at death of the parent and that of the offspring. He arranged the parents into four groups, according to the age at which they died, and the offspring into five groups upon the same basis. In the case of the parents the groups were: First, those dying under 40;

TABLE I

Analysis of the Hyde family data by person's age at death, showing the number and percentage having (a) fathers and (b) mothers who died at the age periods named. (From Bell).

Person's age at death	Father's age at death				
	Stated	-40	40-60	60-80	80+
Stated	2,287	66	522	1,056	643
Under 20	669	20	189	299	161
20 and under 40	538	18	140	269	111
40 and under 60	467	12	116	215	124
60 and under 80	428	13	57	196	162
80 and over	185	3	20	77	85
Percentages					
Stated	100.0	2.9	22.8	46.2	28.1
Under 20	100.0	3.0	28.2	44.7	24.1
20 and under 40	100.0	3.4	26.0	50.0	20.6
40 and under 60	100.0	2.6	24.8	46.0	26.6
60 and under 80	100.0	3.0	13.3	45.8	37.5
80 and over	100.0	1.6	10.8	41.6	46.0

Person's age at death	Mother's age at death				
	Stated	-40	40-60	60-80	80+
Stated	1,805	191	435	713	466
Under 20	511	88	129	199	95
20 and under 40	407	42	104	176	85
40 and under 60	379	27	92	159	101
60 and under 80	360	26	80	129	125
80 and over	148	8	30	50	60
Percentages					
Stated	100.0	10.6	24.1	39.5	25.8
Under 20	100.0	17.2	25.2	39.0	18.6
20 and under 40	100.0	10.3	25.6	43.2	20.9
40 and under 60	100.0	7.1	24.3	42.0	26.6
60 and under 80	100.0	7.2	22.2	35.9	34.7
80 and over	100.0	5.4	20.3	33.8	40.5

second, between 40 and 60; third, between 60 and 80; and fourth, at age 80 and over. The groups for the offspring were the same, except that the first was divided into two parts, namely, those dying under 20 and those dying between 20 and 40. The resulting figures are exhibited in Table 1.

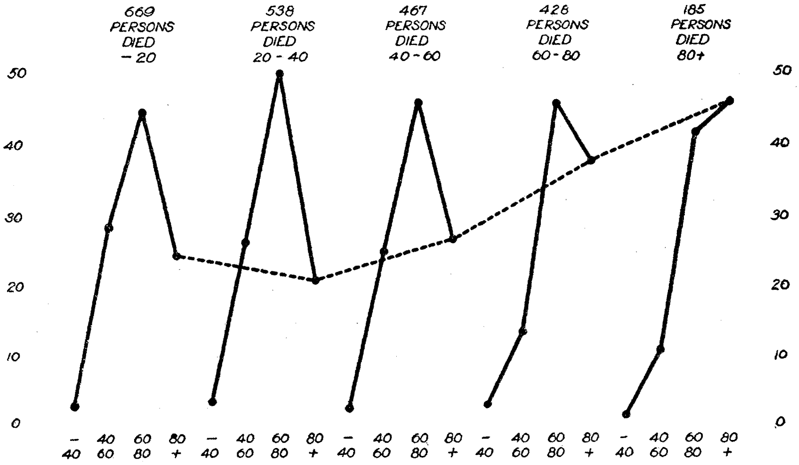

FIG. 2. INFLUENCE OF FATHER'S AGE AT DEATH UPON LONGEVITY OF OFFSPRING. First dot in each diagram shows the percentage having fathers who died at 40; second dot the percentage having fathers who died from 40—60; third dot the percentage having fathers who died from 60—80; fourth dot the percentage having fathers who died 80+

The results for father and offspring are shown in Figure 2, based upon the data of Table 1. In each of the 5 polygons, one for each offspring group, the first dot shows the percentage of fathers dying under 40; the second dot the percentage of fathers dying between 40 and 60; and so on, the last dot in each curve showing the percentage of fathers dying at age 80 and over. It is to these last dots that attention should be particularly directed. It will be noted that the dotted line connecting the last dots of each of the 5 polygons in general rises as we pass from the left-hand side of the diagram to the right-hand side. In the case of offspring dying under 20, 24 per cent. of their fathers died at ages over 80. About 21 per cent. of the fathers of offspring dying between 20 and 40 lived to be 80 years or over. For the next longer-lived group of offspring, dying between 40 and 60, the percentage of fathers living to 80 or over rose to 27 per cent. In the next higher group, the percentage is nearly 38, and finally the extremely long-lived group of offspring, the 185 persons who died at ages of 80 and over, had 46 per cent. or nearly one half of their fathers living to the same great age. In other words, we see in general that the longer-lived a group of offspring is, on the average, the longer-lived are their fathers, on the average; or, put in another way, the higher the percentage of very long-lived fathers which this group will have as compared with shorter-lived individuals.

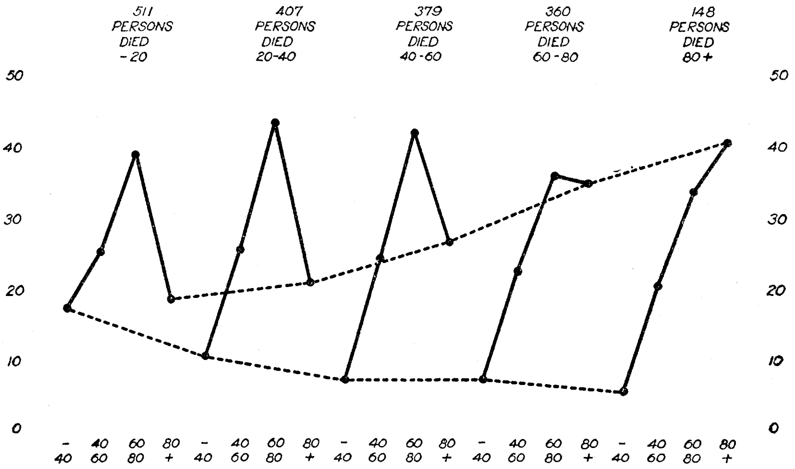

FIG. 3. INFLUENCE OF MOTHER'S AGE AT DEATH UPON LONGEVITY OF OFFSPRING. First dot in each diagrom shows the percentage having mothers who died at 40; second dot the percentage having mothers who died at 40—60; third dot the percentage having mothers who died 60—80; fourth dot the percentage having mothers who died 80+

Figure 3 shows the same sort of data for mothers and offspring. Here we see the curve of great longevity of parents rising in an even more marked manner than was the case with fathers of offspring. The group of offspring dying at ages under 20 had only 19 per cent. of their mothers living to 80 and over, whereas the group of offspring who lived to 80 and beyond had 41 per cent. of their mothers attaining the same great age. At the same time we note from the dotted line at the bottom of the chart that as the average age at death of the offspring increases, the percentage of mothers dying at early ages, namely, under 40, as given by the first dots, steadily *decreases* from 17 per cent. at the first group to just over 5 per cent. for the offspring dying at very advanced ages.

These striking results demonstrate at once that there is a definite and close connection between the average longevity of parents and that of their children. Extremely long-lived children have a much higher percentage of extremely long-lived parents than do shorter lived children. While the diagrams demonstrate the fact of this connection, they do not measure its intensity with as great precision as can be obtained by other methods of dealing with the data. A little farther on we shall take up the consideration of this more precise method of measurement of the hereditary influence in respect of longevity.

In the preceding diagrams we have considered each parent separately in connection with the offspring in regard to longevity. We shall, of course, get precisely the same kind of result if we consider both parents together. For the sake of simplicity, taking only the cases of extreme longevity, namely, persons living to 80 or over—the essential data are given in Table 2.

TABLE 2

Longevity of parents of persons dying at 80 and over. (From Bell).

Age at death of parents	Number of persons	Number of persons lived 80+	Per cent. of persons lived 80+
Stated	1,594	139	8.7
Lived to be 80+			
Neither parent...........	827	44	5.3
One parent (not other)...	583	57	9.8
Both parents.............	184	38	20.6
Father (not mother).....	337	38	11.3
Mother (not father)......	246	19	7.7

From this table it is seen that where neither parent lived to be 80, only 5.3 per cent. of the offspring lived to be 80 or over, the percentage being based upon 827 cases. Where one parent, but not the other, lived to be 80 or older, 9.8 per cent. of the offspring lived to be 80 or older, the percentage here being based upon 583 cases. Where both parents lived to be 80 or older 20.6 of the persons lived to the same great age, the percentage being based upon 184 cases. Thus it appears that in this group of people four times as many attained great longevity if both of their parents lived to an advanced age, as attained this age when neither parent exhibited great longevity. The figures from the Hyde family seem further to indicate that the tendency of longevity is inherited more strongly through the father than through the mother. Where the father, but not the mother, lived to be 80 or older, 11.3 per cent. of the persons lived to age 80 or more, there being 337 cases of this kind. Where the mother, but not the father, lived to be 80 or older, only 7.7 per cent., or nearly 4 per cent. fewer of the persons lived to the advanced age of 80 or more, there being 246 cases of this sort. Too much stress is not, however, to be laid upon this parental difference because the samples after all are quite small.

One other point in this table deserves consideration. Out of the 1,594 cases as a whole, less than 9 per cent. of the persons lived to the advanced age of 80 or more. But out of this number there are 767, or 48.1 per cent., nearly one-half of the whole, who had parents who lived to 80 or more years.

Another interesting and significant way in which one may see the great influence of the age of the parents at death upon the longevity of the offspring, is indicated in Table 3, where we have the average duration of life of individuals whose fathers and mothers died at the specified ages.

TABLE 3

Showing the influence of a considerable degree of longevity in both father and mother upon the expectation of life of the offspring. (After Bell). (In each cell of the table the open figure is the average duration of life of the offspring and the bracketed figure is the number of cases upon which the average is based).

Father's age at death	Mother's age at death		
	Under 60	60-80	Over 80
Under 60	32.8 years (128)	33.4 years (120)	36.3 years (74)
60-80	35.8 (251)	38.0 (328)	45.0 (172)
Over 80	42.3 (131)	45.5 (206)	52.7 (184)

We see that the longest average duration of life, or expectation of life, was of that group which had both mothers and fathers living to age 80 and over. The average duration of life of these persons was 52.7 years. Contrast this with the average duration of life of those whose parents both died under 60 years of age, where the figure is 32.8 years. In other words, it added almost exactly 20 years to the average life of the first group of people to have extremely long-lived parents, instead of parents dying under age 60. In each column of the table the average duration of life advances as we proceed from top to bottom—that is, as the father's age at death increases—and in each row of the table the average expectation of life of the offspring increases as we pass from left to right—that is, with increasing age of the mother at death. However the matter is taken, a careful selection of one's parents in respect of longevity is the most reliable form of personal life insurance.

So much for Bell's analysis of longevity in the Hyde family. We have seen that it demonstrates with the utmost clearness and certainty that there is an hereditary influence between parent and offspring affecting the expectation of longevity of the latter. Bell's method of handling the material does not provide any precise measure of the intensity of this hereditary influence, nor does it furnish any indication of its strength in any but the direct line of descent. Of course, if heredity is a factor in the determination of longevity we should expect its effects to be manifested as between brothers and sisters, or in the avuncular relationships, and in greater or less degree in all the other collateral and more remote direct degrees of kinship. Happily, we have a painstaking analysis, with a quantitative measure of the relative influence of heredity in the determination of longevity, which was carried out many years before Bell's work on the Hyde family, by the pioneer in this field, Prof. Karl Pearson. His demonstration of the inheritance of longevity appeared more than twenty years before that of Bell. I have called attention to the latter's work first merely because of the greater simplicity and directness of his demonstration. We may now turn to a consideration of Pearson's more detailed results.

3. Pearson's Work

The material used by Pearson and his student, Miss Beeton, who worked with him on the problem, came from a number of different sources. Their first study dealt with three series from which all deaths recorded as due to accident were excluded. The first series included one thousand cases of the ages of fathers and sons at death, the latter being over 22.5 years of age, taken from Foster's "Peerage." The second series consisted of a thousand pairs of fathers and sons, the latter dying beyond the age of 20, taken from Burke's "Landed Gentry." The third series consisted of ages at death of one thousand pairs of brothers dying beyond the age of 20 taken from the "Peerage." It will be noted that all these series considered in this first study dealt only with inheritance in the male line. The reason for this was simply that in such books of record as the "Peerage" and "Landed Gentry" sufficiently exact account is not given of the deaths of female relatives. In a second study the material was taken from the pedigree records of members of the English Society of Friends, and from the Friends Provident Association. This material included data on inheritance of longevity in the female line and also provided data for deaths of infants, which were lacking in the earlier used material. The investigation was grounded upon that important branch of modern statistical calculus known as the method of correlation. For each pair of relatives between whom it was desired to study the intensity of inheritance of longevity a table of double entry was formed, like the one shown here as Table 4.

TABLE 4

Correlation table showing the correlation between father and son in respect of duration of life.

Duration of Life of Father

		23	28	33	38	43	48	53	58	63	68	73	78	83	88	93	98	103	Totals
Duration of Life of Son	23	1	1	2	5	3	11	6	7	11	9	6	12	8	2		2		86
	28				1	6	4	5	12	15	10	13	10	7		1	1		85
	33			1	2	2	5	7	8	7	10	7	8	8	4	1			70
	38		1	1	2		2	8	5	3	9	11	11	9	5	2	1		70
	43		1			1	5	1	5	6	11	10	10	17	5				72
	48			1	1	2	5	5	4	6	9	12	15	5	3				68
	53		1		3		5	7	3	2	11	11	14	10	1	1	1		70
	58			1	3		4	5	10	8	10	5	8	9	3		2		68
	63		2	1	3	5	1	4	8	13	9	11	11	11	5				84
	68			1	6	3	6	7	5	5	6	14	16	12	7	2			90
	73		1		2	1	6	5	4	7	9	10	14	13	8	8	1	1	90
	78			1	1	2	2	4	4	4	10	5	8	9	4		3		57
	83				1	1	5	3	1	2	3	7	10	13	3	2	2		53
	88		1				2	3		1	4	7	5	1	2		2		28
	93						1				2	2							5
	98						1						1		1	1			4
Totals		1	8	9	30	26	65	70	76	90	122	131	153	132	53	18	15	1	1000

The figures in each cell or compartment of this table denote the frequency of occurrence of pairs of fathers and adult sons having respectively the durations of life indicated by the figures in the margins. Thus we see, examining the first line of the table, that there were 11 cases in which the average duration of life of the father was 48 years and that of the adult son 23 years. Farther down and to the right in the table there were 13 cases in which the average duration of life of the father and the son was in each case 83 years. These cases are mentioned merely as illustrations. The whole table is to be read in the same manner.

From such a table as this it is possible to calculate, by well-known mathematical methods, a single numerical constant of somewhat unique properties known as the coefficient of correlation, which measures the degree of association or mutual dependence of the two variables included in such double entry tables. This coefficient measures the amount of resemblance or association between characteristics of individuals or things. It is stated in the form of a decimal which may take any value between 0 and 1. As the correlation coefficient rises to 1 we approach a condition of absolute dependence of the variables one upon the other. As it falls to zero we approach a condition of absolute independence, where the one variable has no relation to the other in the amount or direction of its variation. The significance of a correlation coefficient is always to be judged, in any particular case, by the magnitude of a constant associated with it called the probable error. A correlation coefficient may be regarded as certainly significant when it has a value of 4 or more times that of its probable error, which is always stated after the coefficient with a combined plus and minus sign between the two. The coefficient is probably significant when it has a value of not less than 3 times its probable error. By "significant" in this connection is meant that the coefficient expresses true organic relationship and not merely a random chance result.

In Table 5 are the numerical results from the first study based upon the "Peerage" and "Landed Gentry."

TABLE 5

Inheritance of duration of life in male line. Data from "Peerage" and "Landed Gentry." (Beeton and Pearson).

Relatives		Correlation coefficient	Ratio of coefficient to its probable error.
x	y	r_{xy}	$r_{xy} \div E_r$
Father ("Peerage")	Son, 25 years and over	.115 ± .021	5.5
Father ("Landed Gentry")	Son, 20 years and over	.142 ± .021	6.8
Father ("Peerage")	Son, 52.5 years and over	.116 ± .023	5.0
Father ("Landed Gentry")	Son, 50 years and over	.113 ± .024	4.7
Brother (Peerage")	Brother	.260 ± .020	13.0

It is seen at once that all of the coefficients are significant in comparison with their probable errors. The last column of the table gives the ratio of the coefficient to its probable error, and in the worst case the coefficient is 4.7 times its probable error. The odds against such a correlation having arisen from chance alone are about 655 to 1. Odds such as these may be certainly taken as demonstrating that the results represent true organic relationship and not mere chance. All of the other coefficients are certainly significant, having regard to their probable errors. Furthermore, they are all positive in sign, which implies that a variation in the direction of increased duration of life in one relative of the pair is associated with an increase in expectation of life in the other. It will be noted that the magnitude of the correlation between brother and brother is about twice as great as in the case of correlation of father with son. From this it is provisionally concluded that the intensity of the hereditary influence in respect of duration of life is greater in the fraternal relationship than in the parental. It evidently makes no difference, broadly speaking, so far as these two sets of material are concerned, whether there are included in the correlation table all adult sons, whatever their age, or only adult sons over 50 years of age. The coefficients in both cases are essentially of the same order of magnitude.

Perhaps some one will be inclined to believe that the correlation between father and son, and brother and brother, in respect of the duration of life arises as a result of similarity of the environments to which they are exposed. Pearson's comments on this point are penetrating, and I believe absolutely sound. He says:

> There may be some readers who will be inclined to consider that much of the correlation of duration of life between brothers is due to there being a likeness of their environment, and that thus each pair of brethren is linked together and differentiated from the general population. But it is difficult to believe that this really affects *adult* brothers or a father and his adult offspring. A man who dies between 40 and 80 can hardly be said to have an environment more like that of his brother or father, who died also at some such age, than like any other member of the general population. Of course, two brothers have usually a like environment in infancy, and their ages at death, even if they die adults, may be influenced by their rearing. But if this be true, we ought to find a high correlation in ages at death of brethren who die as minors. As a matter of fact this correlation for minor and minor is 40 to 50 per cent. less than in the case of adult and adult. It would thus seem that identity of environment is not the principal factor in the correlation between ages of death, for this correlation is far less in youth than in old age.

The results regarding minors to which Pearson refers are shown in Table 6. This table gives the results of the second study made by Beeton and Pearson on inheritance of duration of life, based upon the records of the Friends Societies. It appears in the upper half of the table that wherever a parent, father or mother, appears with a minor son or daughter the correlation coefficients are small in magnitude. In some cases they are just barely significant in comparison with their probable errors, as for example, the correlation of father and minor

TABLE 6

Inheritance of duration of life. Data from Quaker records.
(Beeton and Pearson).

Relatives		Correlation coefficient	Ratio of coefficient to its probable error.
x	y	r_{xy}	$r_{xy} \div E_r$
Father	Adult son	0.135 ± .021	6.4
Father	Minor son	.087 ± .022	4.0
Father	Adult daughter	.130 ± .020	6.5
Father	Minor daughter	.052 ± .023	2.3
Mother	Adult son	.131 ± .019	6.9
Mother	Minor son	.076 ± .024	3.2
Mother	Adult daughter	.149 ± .020	7.5
Mother	Minor daughter	.138 ± .024	5.7
Elder adult brother	Younger adult brother	.229 ± .019	12.1
Adult brother	Adult brother	.285 ± .020	14.3
Minor brother	Minor brother	.103 ± .029	3.6
Adult brother	Minor brother	-.026 ± .025	1.0
Elder adult sister	Younger adult sister	.346 ± .018	19.2
Adult sister	Adult sister	.332 ± .019	17.5
Minor sister	Minor sister	.175 ± .031	5.6
Adult sister	Minor sister	-.026 ± .029	.9
Adult brother	Adult sister	.232 ± .015	15.5
Minor brother	Minor sister	.144 ± .025	5.8
Adult brother	Minor sister	-.006 ± .035	.2
Adult sister	Minor brother	-.027 ± .024	1.1

The cases above the horizontal line are all direct lineal inheritance; those below the line collateral inheritance.

son, and that of mother and minor daughter. In the other cases involving minors the coefficients are so small as to be insignificant. On the other hand, in every case of correlation between parent and *adult* offspring of either sex, the coefficient is 6 or more times its probable error, and must certainly be regarded as significant. It will further be noted that the magnitude of the coefficients obtained from these Quaker records is of the same general order as was seen in the previous table based on the "Peerage" and "Landed Gentry" material.

The lower part of the table gives the results for various fraternal relationships. In general the fraternal correlations are higher than the parental. The coefficients for minors or for minors with adults are very low and in most cases not significantly different from zero.. In four cases—namely, adult brother with minor brother; adult sister with minor sister; adult brother with minor sister; and adult sister with minor brother—the coefficients are all negative in sign, although in no one of the cases is the coefficient significant in comparison with its probable error. A minus sign before a correlation coefficient means that an increase in the value of one of the variables is associated with a decrease in the value of the other. So that these negative cofficients would mean, if they were significant, that the greater the age at death of an adult brother, the lower the age at death of his minor brother or sister. But the coefficients are actually sensibly equal to zero. Pearson points out that the minus sign in the case of these correlations of adult with minor exhibits the effect of the inheritance of the mortality of youth. Minors dying from 16 to 20 are associated with adults dying from 21 to 25. That is, minors dying late correspond to adults dying early. This situation may be a peculiarity of the Quaker material with which this work deals. There is urgent need for further study of the inheritance of the duration of life on more and better material than any which has hitherto been used for the purpose. I have under way in my own laboratory at the present time an extensive investigation of this kind, in which there will be hundreds of thousands of pairs of relatives in the individual correlation tables instead of thousands, and all types of collateral kinship will be represented. Because of the magnitude of the investigation, however, it will be still a number of years before the results will be in hand for discussion.

The facts which have been presented leave no doubt as to the reality of the inheritance factor as a prime determinant of the length of the life span.

At the beginning it was pointed out that it was on *a priori* grounds highly probable that duration of life is influenced by both heredity and environment, and that the real problem is to measure the comparative effect of these two general sets of factors. We have seen that the intensity of inheritance of duration of life, taking averages, is of the order indicated by the following coefficients.

Parental correlation (adult children) $r = .1365$
Fraternal correlation (adults) $r = .2831$

Now we have to ask this question: What are the values of parental and fraternal correlation for characters but slightly if at all affected in their values by the environment? Happily, Pearson has provided such values in his extensive investigations on the inheritance of physical characters in man.

TABLE 7

Parental inheritance of physical characters in man. (Pearson).

Pair	Organ	Correlation
Father and Son	Stature	.51
" " "	Span	.45
" " "	Forearm	.42
" " "	Eye Color	.55
Father and Daughter	Stature	.51
" " "	Span	.45
" " "	Forearm	.42
" " "	Eye Color	.44
Mother and Son	Stature	.49
" " "	Span	.46
" " "	Forearm	.41
" " "	Eye Color	.48
Mother and Daughter	Stature	.51
" " "	Span	.45
" " "	Forearm	.42
" " "	Eye Color	.51

In Table 7 are given the values of the parental correlations for the four physical characters—stature, span, forearm length, and eye color. Now it is obvious that the differences of environmental forces impinging upon the various members of a homogeneous group of middle class English families (from which source the data for these correlations were drawn) can by no possibility be great enough to affect sensibly the stature, the arm-length, or the eye color of the adults of such families. It would be preposterous to assert that the resemblance between parents and offspring in respect of eye color is due solely, or even sensibly, to similarity of environment.

It is due to heredity and substantially nothing else. Now the average value of the 16 parental coefficients for the inheritance of physical characters shown in the table is

$$r = .4675$$

TABLE 8

Fraternal inheritance of physical characters in man. (*Pearson*).

Pair	Organ	Correlation
Brother and Brother	Stature	.51
" " "	Span	.55
" " "	Forearm	.49
" " "	Eye color	.52
" " "	Cephalic index	.49
" " "	Hair color	.59
Sister and Sister	Stature	.54
" " "	Span	.56
" " "	Forearm	.51
" " "	Eye color	.45
" " "	Cephalic index	.54
" " "	Hair color	.56
Brother and Sister	Stature	.55
" " "	Span	.53
" " "	Forearm	.44
" " "	Eye color	.46
" " "	Cephalic index	.43
" " "	Hair color	.56

Table 8 shows the coefficients for the fraternal inheritance of six physical characters, cephalic index (the ratio of head length and head breadth) and hair colour having been added to those given in the parental table. Again it is seen that the coefficients have all about the same values, and it is as apparent as before that the resemblance between brother and sister, for example, in eye-color, or arm length, or shape of head can not for a moment, because of the nature of the characters themselves, be supposed to have arisen because of the similarity of environment. The average value of all these fraternal coefficients is

$$r = .5156$$

From these data, with the help of a method due to Pearson, it is possible to determine the percentage of the death rate dependent upon the inherited constitution, and the percentage not so dependent. If

pN be the number of deaths in N cases which depend in no way upon the inherited constitution of the individual, then $(1\text{-}p)$ will represent the chance of an individual dying because of his inherited constitutional makeup, and $(1\text{-}p)^2$ will be the chance of a pair of individuals, say two brothers, both dying from causes determined by inheritance. If further r denotes the observed correlation between individuals in respect of duration of life, and r_0 the correlation between the same kin in respect of such measured physical characters as those just discussed, in the determination of which it is agreed that environment can play only a small part, we have the following relation:

$$\frac{r}{r_0} = (1\text{-}p)^2$$

Substituting the ascertained values we have

1. From parental correlations.

$$0.1365 = .4675\ (1\text{-}p)^2$$
$$(1\text{-}p)^2 = .292$$
$$(1\text{-}p) = .54$$

2. From fraternal correlations

$$0.2831 = .5156\ (1\text{-}p)^2$$
$$(1\text{-}p) = .74$$

From these figures it may be concluded, and Pearson does so conclude, that from 50 to 75 per cent. of the general death rate within the group of the population on which the calculations are based, is determined fundamentally by factors of heredity and is not capable of essential modification or amelioration by any sort of environmental action, however well intentioned, however costly, or however well advertised. *Mutatis mutandis* the same conclusion applies to the duration of life. I have preferred to state the conclusion in terms of death rates because of the bearing it has upon a great deal of the public health propaganda so loosely flung about. It need only be remembered that there is a perfectly definite functional relation between death rate and average duration of life in an approximately stable population group, expressible by an equation, in order to see that any conclusion as to the relative influence of heredity and environment upon the general death rate must apply with equal force to the duration of life.

4. THE SELECTIVE DEATH RATE IN MAN

If the duration of life were inherited it would logically be expected that some portion of the death rate must be selective in character. For inheritance of duration of life can only mean that when a person dies is in part determined by that individual's biological constitution or makeup. And equally it is obvious that individuals of weak and unsound constitution must, on the average, die earlier than those of strong, sound, and vigorous constitution. Whence it follows that the chances of leaving offspring will be greater for those of sound constitution than for the weaklings. The mathematical discussion which has just been given indicates that from one-half to three-fourths of the death rate is selective in character, because that proportion is determined by hereditary factors. Just in proportion as heredity determines the death rate, so is the death rate selective. The reality of the fact of a selective death rate in man can be very easily shown graphically.

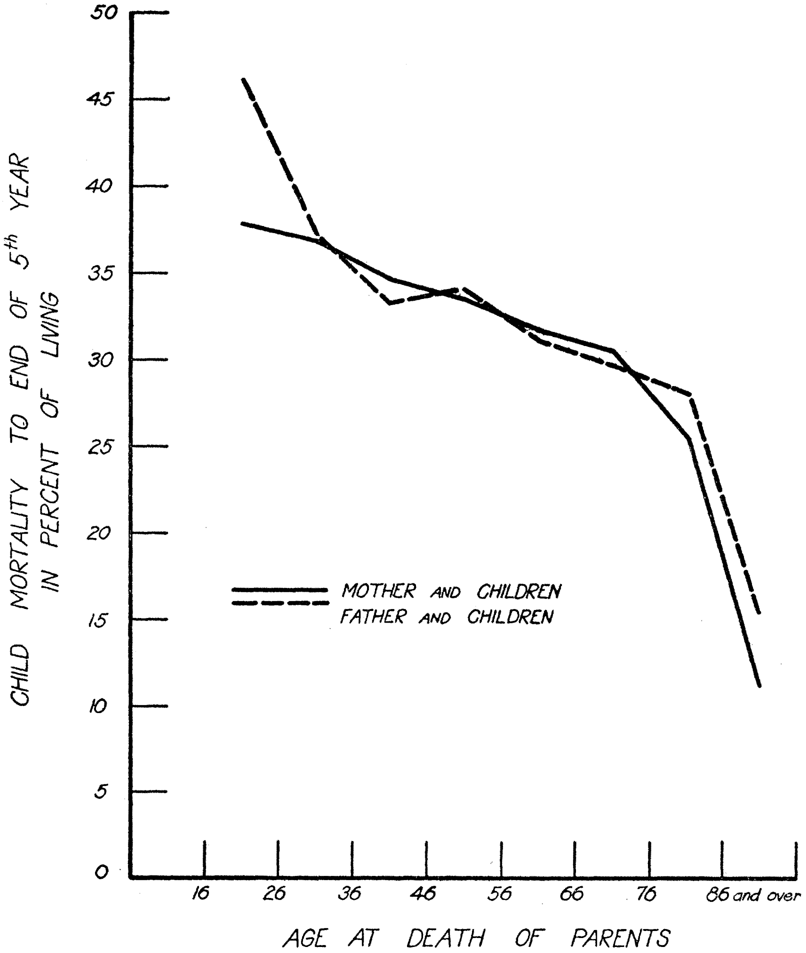

FIG. 4. DIAGRAM SHOWING THE INFLUENCE OF AGE AT DEATH, PARENTS UPON THE PERCENTAGE OF OFFSPRING DYING UNDER 5 YEARS. (After Ploetz)

In Fig. 4 are seen the graphs of some data from European royal families, where no neglect of children, degrading environmental conditions, or economic want can have influenced the results. These data were compiled by the well-known German eugenist, the late Professor Ploetz of Munich. The lines show the falling percentage of the infantile death rate as the duration of life of the father and mother increases. Among the children of short-lived fathers and mothers, at the left end of each line, is found the highest infant mortality, while among the offspring of long-lived parents the lowest infant mortality occurs, as shown at the righthand end of the diagram.

The results so far presented regarding a selective death rate and inheritance of duration of life, have come from selected classes; the aristocracy, royalty or Quakers. None of these classes can be fairly said to represent the general population. Can the conclusion be transferred safely from the classes to the masses? To the determination of this point one of Pearson's students, Dr. E. C. Snow, addressed himself. The method which he used was, from the necessities of the case, a much more complicated and indirect one than that of Pearson and Ploetz. Its essential idea was to see whether infant deaths weeded out the unfit and left as survivors the stronger and more resistant. All the infants born in a single year were taken as a cohort and the deaths occurring in this cohort in successive years were followed through. Resort was had to the method of partial or net correlation. The variables correlated in the case of the Prussian data were these:

1. x_0 = Births in year a given cohort started.
2. x_1 = Deaths in the first two years of life.
3. x_2 = Deaths in the next eight years of life.
4. x_3 = Deaths in the ten years of all individuals not included in the particular cohort whose deaths are being followed.

In the case of the English data the variables were:

x_0 = Births in specified year.
x_1 = Deaths in the first three years of life of those born in specified year.
x_2 = Deaths in fourth and fifth years of life of those born in specified year.
x_3 = The "remaining" deaths under 5.

The underlying idea was to get the partial or net correlation between x_1 and x_2, while x_0 and x_3 are held constant. If the mortality of infancy is selective, its amount should be negatively correlated to a significant degree with the mortality of the next eight years when the births in each district considered are made constant and when the general health environment is made constant. Under the constant conditions specified a negative correlation denotes that the heavier the infantile death rate in a cohort of births the lighter will be the death rate of later years, and *vice versa.* The last variable, x_3, is the one chosen, after careful consideration and many trials, to measure variation in the health environment. If any year is a particularly unhealthy one—an epidemic year for example—then this unhealthiness should be accurately reflected in the deaths of those members of the population not included in the cohort under review.

Snow's results for English and Prussian rural districts are set forth

TABLE 9

Snow's results on selective death rate in man. English and Prussian rural districts.

Data		Actual correlation $r_{12 \cdot 03}$	Expected correlation if no selection
Males:			
English Rural Districts	(1870)	−0.4483	−0.0828
	(1871)	− .3574	− .1014
	(1872)	− .2271	− .0807
Prussian Rural Districts	(1881)	− .9278	− .0958
	(1882)	− .6050	− .0765
Females:			
English Rural Districts	(1870)	− .4666	− .0708
	(1871)	− .2857	− .0505
	(1872)	− .5089	− .0496
Prussian Rural Districts	(1881)	− .8483	− .0933
	(1882)	− .6078	− .0705

in Table 9. From this table it is seen that in every case the correlations are *negative*, and therefore indicate that the mortality of early life is selective. Furthermore, the demonstration of this fact is completed by showing that the observed coefficients are from 3 to 10 times as great as they would be if there were no selective character to the death rate. The coefficients for the Prussian population, it will be noted, are of a distinctly higher order of magnitude than those for the English population. This divergence is probably due chiefly to differences in the quality of the fundamental statistical material in the two cases. The Prussian material is free from certain defects inherent in the English data, which cannot be entirely got rid of. The difference in the coefficients for the two successive Prussian cohorts represents, in Snow's opinion, probably a real fluctuation in the intensity of natural selection in the one group as compared with the other. How significant Snow's results are is shown graphically in Figure 5.

Snow's own comments on his results are significant. He says:

> The investigations of this memoir have been long and laborious, and the difficulties presented by the data have been great. Still, the general result cannot be questioned. *Natural selection, in the form of a selective death-rate, is strongly operative in man in the early years of life.* Those data which we believe to be the best among those we have used—the Prussian figures—show very high negative correlation between the deaths in the first two years of life and those in the next eight, when allowance is made for difference in environment. We assert with great confidence that a high mortality in infancy (the first two years of life) is followed by a corresponding low mortality in childhood, and conversely. The English figures do not allow such a comprehensive survey to be undertaken, but, so far as they go they point in the same direction as the Prussian ones. The migratory tendencies in urban districts militate against the detection of selective influences there, but we express the belief that those influences are just as prevalent in industrial

as in rural communities, and could be measured by other means if the data were forthcoming.

Our investigation substantiates for a general population the results found by Pearson and Ploetz for more restricted populations, and disagrees with many statements of health officers. It is with great reluctance that we point out this disagreement, and assert a doctrine which, in the present sentiment of society, is bound to be unpopular. We have no feelings of antagonism towards the efforts which have been made in recent years to save infant life, but we think that the probable consequences of such actions, so far as past experience can indicate them, should be completely understood. All attempts at the reduction of mortality of infancy and childhood should be made in the full knowledge of the facts of heredity. Everybody knows the extreme differences in constitutional fitness which exist in men and women. Few intelligent people can be ignorant of the fact that this constitutional fitness is inherited according to laws which are fairly definitely known. At the same time marriage is just as prevalent among those of weak stocks as

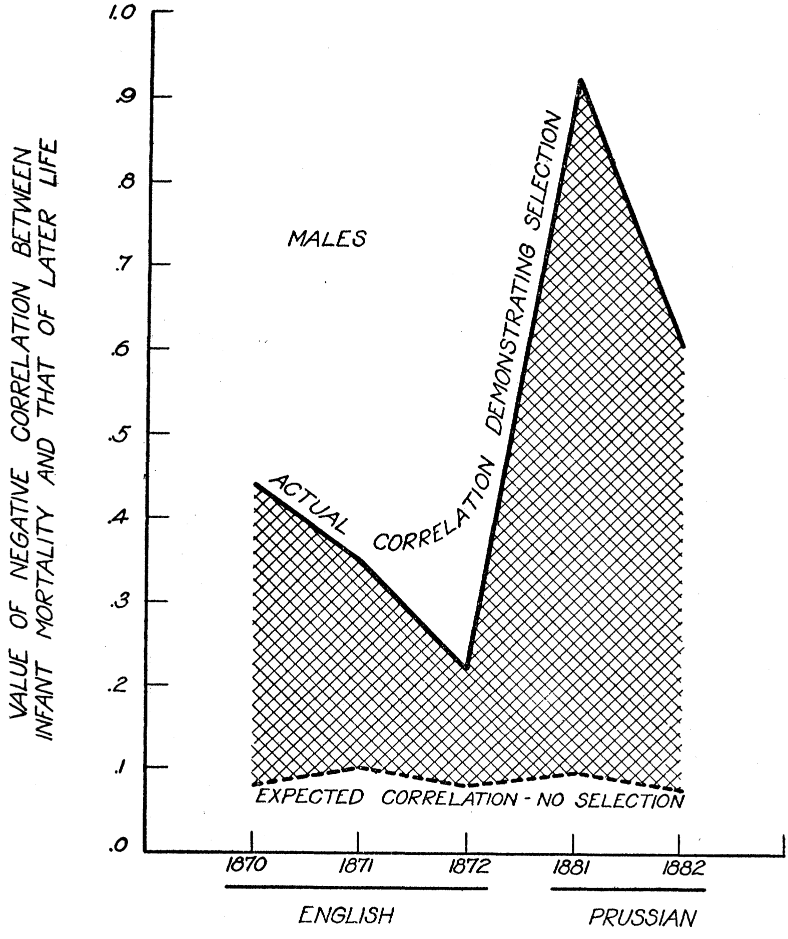

FIG. 5. SNOW'S RESULTS ON SELECTIVE DEATH RATE IN MAN. The cross-hatched area may be taken, in comparison with the small clear area at the bottom, to measure the influence of the selective death rate in increasing the correlations

> among those of the vigorous, while the fertility of the former is certainly not less than that of the latter. Thus a proportion of the infants born every year must inevitably belong to the class referred to in the report as "weaklings," and, with Pearson's results before us, we are quite convinced that true infantile mortality (as distinct from the mortality due to accident, neglect, etc.—no small proportion of the whole) finds most victims from among this class. Incidentally we would here suggest that no investigation into the causes of infant and child mortality is complete until particulars are gathered by the medical officers of the constitutional tendencies and physical characters of the parents.
>
> Our work has led us to the conclusion that infant mortality *does* effect a "weeding out" of the unfit; but, though we would give this conclusion all due emphasis, we do not wish to assert that any effort, however small, to the end of reducing this mortality is undesirable. Nobody would suggest that the difference between the infant rates in Oxfordshire and Glamorganshire (73 and 154 per 1,000 births respectively, in 1908) was wholly due to the constitutional superiority of the inhabitants of the former county. The "weeding-out" process is not uniform. In the mining districts of South Wales, accident, negligence, ignorance and unsanitary surroundings account for much. By causing improvements under these heads it may be possible to reduce the infant mortality of Glamorganshire by the survival of many who are not more unfit than are those who survive in Oxfordshire, and the social instincts of the community insist that this should be done.

This work of Snow's aroused great interest, and soon after its appearance was controverted, as it seems to me quite unsuccessfully, by Brownlee, Saleeby and others.

Happily the results of Pearson, Ploetz and Snow on the selective death rate have recently been accorded a confirmation and extension to still another group of people—the Dutch—in some as yet unpublished investigations carried out by Dr. F. S. Crum of the Prudential Life Insurance Company, with the assistance of the distinguished mathematical statistician, Mr. Arne Fisher. By the kind permission of these gentlemen I am able to state the general results of these investigations in advance of their publication.

The Dutch Government publishes annually data which undoubtedly furnish the best available material now existing in the world for the purpose of determining whether or not there is a positive or negative correlation between infant mortality and the mortality in the immediately subsequent years of life. Fisher's mathematical analysis embraces a very large body of material, including nearly a million and a half births, and nearly a quarter of a million deaths of males occurring in the first five years of life. The Holland data make it possible to develop life tables for every cohort of births and this has been done in the 16 cohorts of males during the years 1901-1916. The data also make it possible to work up these life tables for urban areas and for rural areas. After carefully eliminating secular disturbances the Holland material appears to prove quite conclusively for the rural districts that there is a definite negative correlation, of significant magnitude, between infant mortality and the mortality in the immediately subsequent years of life. The only place where positive correlation appears is in the four large cities of the country with more than a hundred thousand inhabitants each. Fisher makes the following point (in a letter to the present writer) in explanation of these positive correlations. He says:

> The larger cities are better equipped with hospital and clinical facilities than the smaller cities and the rural districts. More money is also spent on child welfare. Is it therefore not possible that many feeble lives who in the course of natural circumstances would have died in the first year of life are carried over into the second year of life by means of medical skill? But medicine cannot always surpass nature, and it might indeed be possible that among cohorts with a low mortality during the first two years of life there will be an increase of death rate in the following three years of life.

Altogether, we may regard the weight of present evidence as altogether preponderant in favor of the view *that the death rate of the earliest period of life is selective—eliminating the weak and leaving the strong.* From our present point of view it adds another broad class of evidential material to the proof of the proposition that inheritance is one of the strongest elements, if not indeed the dominating factor, in determining the duration of life of human beings.